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Coronary Venous Aneurysm in Patients Without Cardiac Arrhythmia as Detected by MDCT

An Anatomic Variant or a Pathologic Entity

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OBJECTIVES The aim of this study was to determine the prevalence of coronary venous aneurysm in patients with no history of cardiac arrhythmia using 64-slice multidetector computed tomography.

BACKGROUND Coronary vein aneurysm frequently has been reported in association with cardiac arrhythmias such as ventricular pre-excitation.

METHODS Coronary computed tomography angiograms of 187 patients (108 men, 79 women; mean age ± SD, 60 ± 12 years) were analyzed retrospectively for the presence of a focal coronary venous aneurysm. Fusiform aneurysm was defined as a focal dilatation of twice the normal vein. However, any size of diverticular aneurysms was included. Cross-sectional diameters of normal and aneurysmal segments of the posterior interventricular vein, great cardiac vein, and coronary sinus (CS) were measured at mid-diastole, late systole, and atrial systole. The Student *t* test was used for continuous variables and contingency tables were used for categorical variables.

RESULTS A single aneurysm was found in 19 (10%) patients (fusiform, n =16; diverticular, n = 3). The most common anatomic location was the posterior interventricular vein near the confluence with the CS (n = 14), followed by the great cardiac vein near the junction with the CS (n = 3), and the CS (n = 2). The mean diameter of the aneurysms was 9.3 ± 1.2 mm (range, 8.1 to 11.4 mm) at mid-diastole and 10.4 ± 1.4 mm (range, 8.5 to 12.7 mm) at late systole. However, the difference was not statistically significant. All normal CSs and 1 aneurysm arising from the CS showed contraction during atrial systole, which may suggest atrial myocardial coverage of these structures. Patients with a venous aneurysm were significantly older than patients without an aneurysm (67.6 ± 11 vs. 59 ± 12 years, respectively; p = 0.006).

CONCLUSIONS Coronary vein aneurysms (especially the fusiform type) were seen in up to 10% of patients with no history of cardiac arrhythmia and can be well visualized on computed tomography angiograms. (J Am Coll Cardiol Img 2010;3:257–65) © 2010 by the American College of Cardiology Foundation

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The coronary venous system consists of a series of tributaries overlying the myocardium, including the coronary sinus (CS), the great cardiac vein (GCV), the anterior interventricular vein, and the posterior interventricular vein (PIV) (Fig. 1) (1–5). Anatomic variation of this system is common (5–7). With increasing use of coronary computed tomography angiography (CTA) to study cardiac anatomic features and the coronary system, knowledge of normal anatomic variations is important to prevent misinterpretation of image findings (7,8).

Coronary vein aneurysm either in the form of a diverticulum or a focal bulbous (fusiform) dilatation has been described with increasing frequency in the literature (2,9–11). These outpouchings may interfere with cannulation of the normal vessel during coronary venous interventions or may cause difficulty at the time of coronary artery bypass surgery (CABG) (12–14). Aneurysms, especially the diverticular form, arising from the CS and the confluence of coronary veins with the CS, may be associated with cardiac arrhythmias such as ventricular pre-excitation resulting from accessory atrioventricular pathways possibly related to muscular continuity between atrial and ventricular myocardium through the outer wall of the venous aneurysm (9–11,15–19). In our practice of reading coronary CTAs, we noted a number of patients whose coronary CTA showed aneurysmal dilatation of the coronary veins. Given that studies have reported on the prevalence of coronary vein aneurysm in patients with pre-excitation syndromes, we decided to determine retrospectively the prevalence of different types of coronary vein aneurysm in a population without a history of arrhythmia. We also measured the cross-sectional diameters of nonaneurysmal and aneurysmal portions of certain coronary veins and compared the results.

ABBREVIATIONS AND ACRONYMS

CABG = coronary artery bypass surgery

CS = coronary sinus

CTA = computed tomography angiography

GCV = great cardiac vein

PIV = posterior interventricular vein

METHODS

The study was conducted with the approval of the institutional review board at the University of California, Irvine, Medical Center and was compliant with Health Insurance Portability and Accountability Act regulations.

Patient selection. Between February and June 2008, we studied 235 consecutive coronary CTAs obtained from 118 patients who were scanned for the assessment of coronary artery disease and from 117 volunteers participating in research studies for which they had given consent. Completed questionnaires on patient clinical histories and their 12-lead electrocardiograms were reviewed, and patients with a history of structural heart disease (n = 7), including cardiomyopathy, congestive heart failure, and valvular heart disease, were removed from the study. Fifteen patients with evidence of cardiac arrhythmia, including atrial fibrillation (n = 11), atrial flutter (n = 2), and ventricular tachycardia (n = 2), also were removed. Seventeen coronary CTAs were excluded because of severe artifacts created by technical problems (n = 2), background noise (n = 4), motion (n = 8), prosthetic heart valves (n = 1), or pacemaker leads (n = 2). Nine studies also were excluded because of poor visualization of the coronary venous system. The remaining (n = 187) examinations (108 men, 79 women; mean age ± SD, 60 ± 12 years) were studied for coronary venous aneurysm.

Computed tomography scan protocol and image reconstruction. Oral and intravenous metoprolol were used to achieve a target heart rate of <65 beats/min as needed. A sublingual nitroglycerin tablet (0.4 to 0.8 mg) was given 1 minute before image acquisition. The mean (± SD) heart rate immediately before data acquisition was 58 ± 5.6 beats/min (range 40 to 73 beats/min). A 64-slice multidetector computed tomography scanner (Toshiba Aquilion, Tustin, California) was used. Contrast enhancement was achieved using 65 to 92 ml (mean 74.91 ± 3.32 ml) of iohexol (Omnipaque 350 mg/ml, Amersham Health, Cork, Ireland) followed by 50 ml saline injected at 4 to 5 ml/sec through an 18-gauge catheter into an antecubital vein. The scan parameters were: collimation, 64 × 0.5 mm; table feed per rotation, 7.2 mm; gantry rotation time, 400 ms; tube voltage, 120 kVP; and tube current, 400 mA. The scan was initiated automatically 4 seconds after reaching a threshold of 180 HU in the descending aorta below the tracheal bifurcation. A retrospective electrocardiography-gated volumetric data set was acquired during a single breath-hold. The mean scan duration was 9.1 ± 1.37 s with a range of 7.4 to 15 s. Using a CTA algorithm, diastolic axial images (thickness, 0.5 mm; increment, 0.3 mm) were reconstructed based on relative-delay strategy at 70%, 75%, and 80% of R-R intervals. A second reconstruction approach was carried out and additional 2-mm data sets were reconstructed at 10% intervals. All the reconstructed data sets were transferred to an offline 3-dimensional workstation (Vital Images, Inc.,

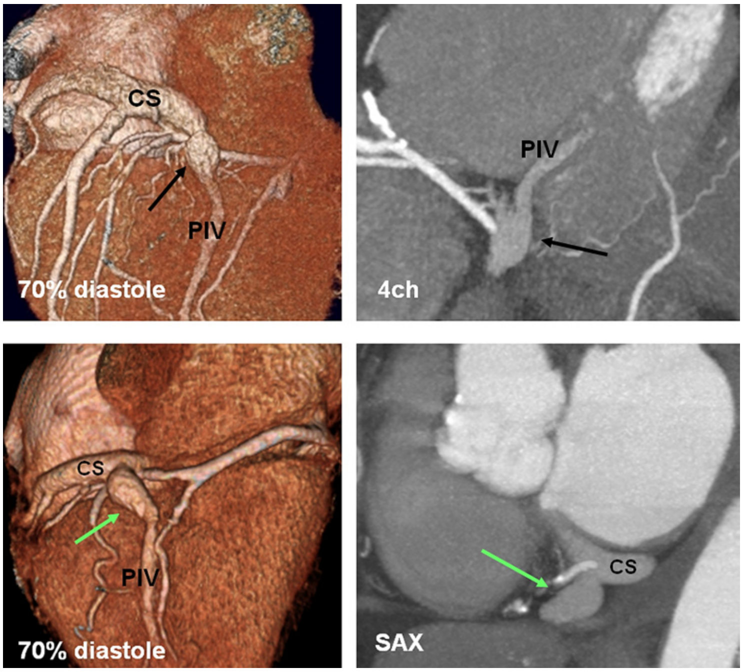

Figure 1. Posterior Interventricular Vein Aneurysm

Posterior interventricular vein (PIV) aneurysm **(black and green arrows)**. Posterior 3-dimensional views and cross-sectional images in 2 asymptomatic patients, a 75-year-old woman **(top row)** and a 74-year-old woman **(bottom row)**. Coronary vein aneurysms usually arise from the PIV near its confluence with the coronary sinus (CS). 4ch = 4-chamber view; SAX = short axis.

Minnetonka, Minnesota) for analysis of the coronary veins.

Computed tomography data analysis and measurements. Multiplanar reformations and 3-dimensional reconstructions of the axial images were rendered and evaluated for the presence of coronary venous aneurysm in consensus by a radiologist and a cardiologist with advanced training in cardiac computed tomography interpretation, as described by the training guidelines of Task Force 12 (20), published in the *Journal of the American College of Cardiology*. Overall image quality was evaluated qualitatively and was classified as excellent, good, or adequate on the basis of primarily common image-degrading artifacts related to metal, motion, or background noise. Patients were grouped according to the presence of a focal venous aneurysm. Each distance was measured twice, and the average value was recorded. All measurements were performed by one author (F.S.) with 17 years experience in computed tomography scanning and 2 research students (S.C. and T.S.) with 2 years experience in cardiac imaging.

The orientation of the focal aneurysm in relation to the long axis of the parent vessel was classified into central (fusiform or bulbous) and eccentric (diverticular or saccular) (2,11). Marginal irregularities and mild focal dilatations are common morphologic variants of veins. However, in the literature, there are no size criteria to label a venous dilatation definitively as an aneurysm. MacDevitt et al. (21) defined a popliteal venous aneurysm as a focal dilatation of twice the normal vein. Therefore, we used this definition for fusiform venous aneurysms on mid-diastolic images. However, all diverticular aneurysms (any size) were included.

After evaluation of the aneurysm shape and location on 3-dimensional and reformatted cross-sectional images, the largest cross-sectional diameter of the aneurysm was measured on images perpendicular to the long axis of the aneurysm. Measurements were obtained at 40%, 70%, and 0% of the cardiac interval. Given an average heart rate of 58 beats/min, we assigned 40% as late-ventricular systole, 70% as mid-ventricular diastole, and 0% as atrial contraction (atrial systole). Cross-sectional diameters of the parent vessels were obtained 1 cm proximal to the aneurysm and the values were averaged. The length of the aneurysm was measured on mid-diastole image series. The relation of the left circumflex artery with the GCV aneurysm was assessed. All coronary CTAs with a diagnosis of

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coronary vein aneurysm were assessed carefully for any associated cardiac or coronary artery anomalies. We assessed the entire venous aneurysm walls for possible attachment with the myocardium of left atrium or ventricles and visually assessed for any change in size of aneurysms during atrial contraction.

To determine whether the size of the parent vessels proximal to the aneurysm were comparable with the size of coronary veins in the group without an aneurysm and to assess possible contraction of the CS and adjacent veins (during atrial systole), we measured the cross-sectional diameters of the coronary veins (CS, GCV, and PIV) in both groups at the above 3 cardiac phases. The average values were used for statistical analysis. Only the diameters of the above 3 vessels were calculated because all aneurysms from our list originated from one of these locations.

The cross-sectional diameters of the CS were measured 2 cm from its ostium and values were averaged. The GCV was measured at the level of a line passing through the mid-portion of the left inferior pulmonary vein. The PIV was measured 1 cm from its confluence with the CS. For cases with PIV aneurysm, cross-sectional diameters of the parent vessels were obtained 1 cm proximal to the aneurysm. In some patients, we also noted the presence of mild varicoid dilatation of a long segment of the PIV. However, we did not categorize this morphologic variant as a focal venous aneurysm. The length and maximum diameter of the varicoid area were measured.

Statistical analysis. Statistical analysis was performed using SAS software version 9.1.3 (SAS Institute, Inc., Cary, North Carolina). Comparisons were performed using the Student unpaired and paired *t* tests for continuous variables. Statistics for all continuous data were reported using the mean ± SD. Chi-square analysis-of-contingency tables were used to compare categoric variables. We report results of the statistical test for each using $p < 0.05$ as the criteria for statistical significance.

RESULTS

Quality of CTA. Image quality was classified as excellent (74.8%; 140 of 187), good (20.9%; 39 of 187), or adequate (4.3%; 8 of 187).

Patients with an aneurysm. A single aneurysm was found in 19 patients (10% of all patients). There were 9 women and 10 men (mean age ± SD, 67.6 ± 11.1 years; range, 47 to 84 years). No patient with multiple aneurysms was identified. Statistical data of venous aneurysms are listed in Table 1. The mean length of the aneurysms at mid-diastole was 13 ± 1.9 mm (range, 9.8 to 17.7 mm). On average, the PIV and GCV fusiform aneurysms exceeded the diameter of the adjacent parent segment by 111% and 132%, respectively, at mid-diastole. Fusiform aneurysms were seen in 16 patients, and the diverticular form was seen in 3 patients (2 from the CS and 1 from the PIV). The most common anatomic location was the PIV at the confluence with the CS (n = 14) (Fig. 1). The second most common location was the GCV near the junction with the CS (n = 3) (Fig. 2). Both aneurysms that originated from the CS were diverticular: one at the origin of the oblique vein of Marshall measuring 17.7 mm in length (Fig. 3) and the second one a diverticulum from the anterior margin of the CS proximal to the confluence of the PIV measuring 14.7 mm in length (Fig. 4). We did not find any aneurysm at other locations. No clear attachment

Table 1. Statistical Data for the Group With Aneurysm

	PIV	GCV	CS	Total
Number	14	3	2	19
Mean age ± SD				67.6 ± 11
Mean aneurysm diameter ± SD (range)				
Late systole	10.5 ± 1.4	9.6 ± 0.6	10.6 ± 2.8	10.4 ± 1.4 (8.5–12.7)
Mid-diastole	9.5 ± 1.2	8.6 ± 0.2	9.6 ± 2.1	9.3 ± 1.2 (8.1–11.4)
Atrial contraction	10.3 ± 1.2	8.5 ± 0.3	8.9 ± 1.1	9.9 ± 1.3 (8.1–12.9)
Mean aneurysm length ± SD (range), mid-diastole	12.5 ± 1.7	12.9 ± 0.3	16.2 ± 2.1	13 ± 1.9 (9.8–17.7)
Mean nonaneurysmal diameter ± SD (range)				
Mid-diastole	4.5 ± 0.8	3.7 ± 0.4	7.9 ± 1.0	4.7 ± 1.4 (3.3–8.7)
Late systole	5.3 ± 1.2	4.5 ± 0.6	8.6 ± 0.1	5.8 ± 1.5 (3.8–8.7)
Atrial contraction	5.8 ± 1.0	4.3 ± 0.4	6.1 ± 0.0	5.6 ± 1.0 (4.0–7.7)

All measurements expressed in millimeters.
CS = coronary sinus; GCV = great cardiac vein; PIV = posterior interventricular vein.

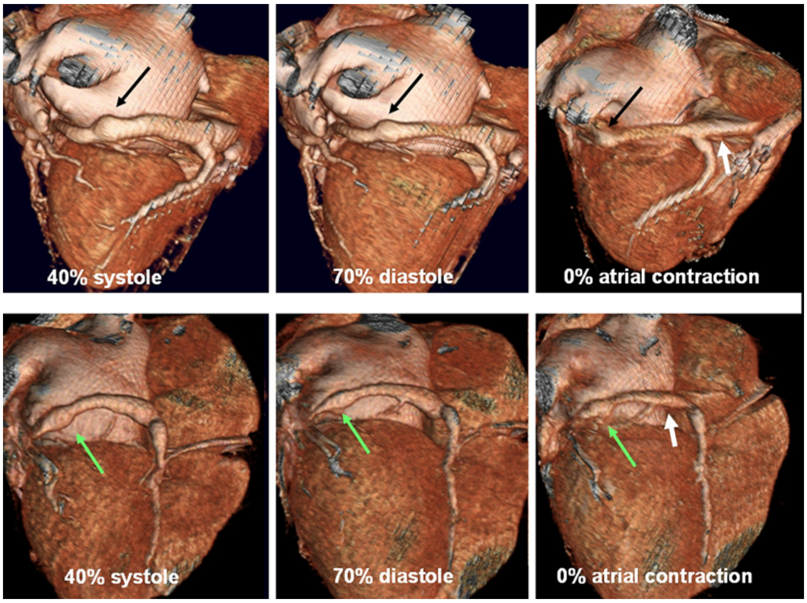

Figure 2. Great Cardiac Vein Aneurysm

Aneurysmal dilatation of the great cardiac vein (GCV) **(black and green arrows)**. Posterior 3-dimensional images of the heart in 2 different patients, an 82-year-old woman **(top row)** and a 70-year-old woman **(bottom row)**. In all patients with a GCV aneurysm, the aneurysm originated from the distal GCV, where it approaches the coronary sinus. The left circumflex artery passed underneath the GCV in all patients with a GCV aneurysm. Images were obtained in late systole (40%), mid-diastole (70%), and atrial contraction (0%). Note that both normal veins and aneurysms are larger in 40% images compared with 70% images. Also note the contraction of the coronary sinus at 0% **(white arrows)**. This is the result of continuity of the right atrial myocardium over the coronary sinus.

was found between the aneurysm wall and the left atrium or ventricles. No contraction of aneurysms was seen during atrial (0%) or ventricular (40%) systole except for the diverticulum arising from the CS at origin of the vein of Marshall, which showed contraction during atrial systole, which may suggest the presence of myocardial fibers in its wall (Fig. 3). In patients with a GCV aneurysm, the left circumflex artery passed anterior to the vein in all 3 patients with no significant compressive effect on the vein.

The mean cross-sectional diameter of the aneurysms was greater in ventricular systole compared with ventricular diastole (approximately a 10% change between the 2 phases). However, the difference was not statistically significant (Table 2) (PIV, $p = 0.0537$; GCV, $p = 0.114$; CS, $p = 0.771$). All patients were asymptomatic at the time of scan. No

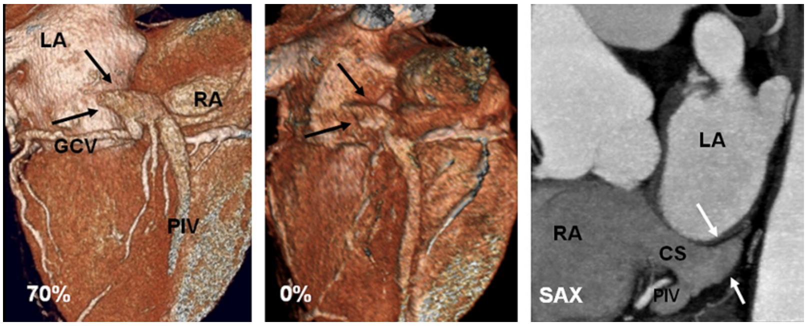

Figure 3. Coronary Sinus Diverticular Aneurysm: Oblique Vein of Marshall Origin

Diverticular aneurysm of the coronary sinus (CS) **(black and white arrows)**. Posterior 3-dimensional views at mid-diastole (70%) and atrial contraction (0%), and short axis (SAX) image at 70% in a 71-year-old man. Contraction of the aneurysm and the CS is shown at 0%, indicating myocardial coverage of these structures. Patient was asymptomatic, but coronary arteries showed moderate disease involving left coronary branches. GCV = great cardiac vein; LA = left atrium; PIV = posterior interventricular vein; RA = right atrium.

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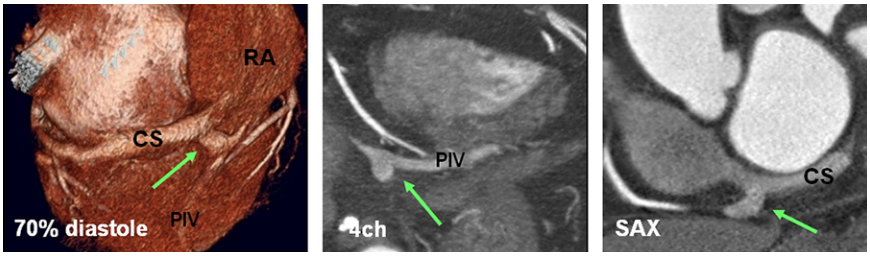

Figure 4. Coronary Sinus Diverticular Aneurysm Near the Posterior Interventricular Vein

Diverticular aneurysm **(green arrows)** arising from the junction of the coronary sinus (CS) and posterior interventricular vein (PIV). Abbreviations as in Figures 1 and 3.

coronary artery anomalies were seen. However, 4 patients (21%) had a history of CABG (3 with PIV aneurysm and 1 with GCV aneurysm), which was significantly higher than the group without an aneurysm (n = 8; 4.7%) (p = 0.0225), suggesting a possible traumatic cause in these patients. However, we did not find any surgical coronary artery anastomosis near the venous aneurysm.

Patients without an aneurysm. This group (n = 168) consisted of 70 women and 98 men (mean age ± SD, 59 ± 12.2 years; range, 27 to 86 years), with no significant gender difference with the aneurysm group (p = 0.633). However, patients in the aneurysm group were significantly older than patients in the group without an aneurysm (p = 0.006). Average cross-sectional cardiac vein diameter in the group without an aneurysm is listed on Table 2. There was no statistically significant difference between the diameter of cardiac veins measured in the group without aneurysm with the diameter of the nonaneurysmal portion of the veins (parent vessel) in the aneurysm group at mid-diastole (PIV: 4.3 vs. 4.5 mm, respectively, p = 0.348; GCV: 4.2 vs. 3.7 mm, respectively, p = 0.142; and CS: 9.2 vs. 7.9 mm, respectively, p = 0.368).

Measurements showed contraction of the CS (a percent change of 18% in diameter size) and no contraction of the PIV and GCV during atrial systole compared with the mid-diastolic phase. Varicoid dilatation of the PIV was seen in 3 patients (2 women and 1 man; mean age ± SD, 67.6 ± 12 years) with a maximum dilatation of 6.3 ± 0.6 mm and average length of 3.7 ± 2.9 cm (Fig. 5).

DISCUSSION

The CS is increasingly used for examining left atrial activity during electrophysiology studies and catheter ablation or as a conduit for left ventricular pacing (12,13). Anatomic variants of the coronary veins, including large valves, anomalous venous return, aneurysms, or atresia, are common (6,22–24). These variants may interfere with catheterization of the target vein and in some cases can be a potential source of cardiac arrhythmias (11,15,16). In recent years, there has been a growing recognition between the association of coronary vein aneurysms with accessory pathways. This is especially true when the bypass tract is located in the posteroseptal region (11,16,18). Muscular continuity between atrial and ventricular myocardium through the outer wall of the venous aneurysm has been proposed as a mechanism for ventricular preexcitation (11–13,25). However, in our patients who had no history of cardiac arrhythmia, we did not identify obvious attachments between the aneurysm wall and the wall of ventricle to indicate possible myocardial continuity.

It is difficult to estimate the true incidence of coronary vein aneurysms, especially in asymptom-

Table 2. Average Cross-Sectional Diameter of Cardiac Veins With Percent Change at 2 Cardiac Phases in the Group Without Aneurysm

Veins (n = 168)	Late Systole Diameter	Mid-Diastole Diameter	Atrial Contraction Diameter	Percent Change From Systole to Mid-Diastole	Percent Change From Mid-Diastole to Atrial Contraction
Posterior interventricular vein	5.2 ± 1.4 (2.6–9.7)	4.3 ± 1.1 (2.4–8.9)	4.3 ± 1.1 (1.8–7.2)	−18%	0%
Great cardiac vein	5.0 ± 1.2 (2.7–10.2)	4.2 ± 1.0 (2.3–8.5)	4.2 ± 1.0 (2.1–7.4)	−16%	0%
Coronary sinus	10.6 ± 2.1 (5.9–20)	9.2 ± 1.9 (4.4–17.0)	7.5 ± 1.6 (4.0–15.0)	−13%	−18%

All measurements expressed as mean ± SD (range) in millimeters.

Figure 5. Varicoid Dilatation of the Posterior Interventricular Vein

Varicoid dilatation of the posterior interventricular vein in 3 different patients. Posterior 3-dimensional views of the heart at mid-diastole (70%).

atic patients. In anatomic studies of human heart by von Lüdinghausen (2), fusiform (bulbous) enlargement of the PIV has been reported in 19% of specimens. Otherwise, most of the reported cases have been diagnosed during coronary venous angiography or transesophageal echocardiography and in patients with posteroseptal and left posterior accessory pathways (11,16–19). In the largest series reported by Sun et al. (11), coronary vein angiography in patients with posteroseptal and left posterior accessory pathways revealed a CS diverticulum in 7.5% (36 of 480) and fusiform enlargement of the small cardiac vein, middle cardiac vein, or CS in 2% (n = 15) of these patients. The median width of the CS diverticulum was 18 mm, but no size was reported for fusiform aneurysms. In another series (19), 10% of patients with posterior septal accessory pathways showed a CS diverticulum (2 to 5 cm in size) during surgery. In our study, fusiform aneurysms were seen in 16 (8.5%) patients and the diverticular form was seen in 3 (1.5%) patients, and the mean length of the aneurysms was 13 mm (16 mm for CS diverticulum). It is possible, then, that larger and diverticular forms of aneurysms may have a higher rate of associated arrhythmias than simple fusiform dilatation, and pathophysiologic mechanisms of development may differ.

Mechanism of development of venous aneurysms. The exact mechanism of venous aneurysm formation is not obvious. However, both acquired and congenital factors may play a role (9,11,25). Diverticula most likely are congenital, whereas fusiform dilatations are probably acquired or merely an anatomic variant. Abnormal embryologic development of the sinus venosus is suggested as the cause of CS aneurysms (25). Associated congenital anomalies have been reported in patients with diverticula of the coronary veins (26). However, this was not apparent in the present report. Intimal hyperplasia and loss of smooth muscle and elastic tissue with replacement by connective tissue are seen in peripheral veins with age (27,28). This may explain why, in the present study, patients with a venous aneurysm were significantly older than patients without an aneurysm. Trauma and hemodynamic modification after coronary surgery also may play a role, because 21% of our patients with an aneurysm had a history of CABG. The CS is surrounded by a myocardial sleeve that continues into the right atrium (29). This myocardial sleeve is thin and difficult to resolve with current computed tomography scans. However, simultaneous contraction of the CS (and one of the CS aneurysms) and the atria during atrial systole shown in our study validates the continuity of right atrial myocardium over the CS and possibly over the CS aneurysm. The CS sleeve normally may extend shortly beyond the orifices of the entering coronary veins, where it thickens in a sphincter-like fashion (myocardial cuff) (29). One explanation for the development of fusiform aneurysms may reside in changing mechanical forces at the level of abrupt termination of the CS myocardial muscle sleeve where it connects with the PIV, GCV, or posterior veins. The cause of fusiform enlargement of the PIV is attributed to the presence of an ectopic venous valve proximal to the aneurysm (2).

Anatomic location of the aneurysms. Most coronary vein aneurysms are located along the inferoseptal aspect of the CS near the confluence of the PIV and usually within 1.5 cm from the CS ostium (11,16). This is consistent with our findings, in which 79% (n = 15) of aneurysms were located at or near the junction of the CS and PIV. Newer technologies, such as multidetector computed tomography, pro-

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vide more anatomic detail, which allows us to differentiate clearly between these aneurysms. In some cases, multiple diverticula may exist. Davidson et al. (25) reported a case of multiple CS diverticula associated with an accessory pathway. We did not see this variant.

In the diagnosis of CS diverticulum, it is important to differentiate this from anatomic variants of the right atrium, especially from the sub-Thebesian recess, which is an outpouching of the right atrial wall at the level of the cavotricuspid isthmus extending below the orifice of the CS (30,31).

The junction of the GCV with the CS is the second most common location of coronary vein aneurysm. The GCV originates at the lower third of the anterior interventricular sulcus and merges with the CS at the posterior atrioventricular sulcus (32,33). The GCV crosses the branches of the left anterior descending and left circumflex arteries. The relation of the vein and these arteries is variable and unpredictable in at least 30% of the cases (4,33). Compression of the vein by a crossing artery has been suggested as a reason for development of a GCV aneurysm. Loukas et al. (14) reported a case of GCV aneurysm at the time CABG surgery where the left circumflex artery crossed the GCV. Their patient had no history of cardiac arrhythmias. We found 3 patients with GCV aneurysm. However, we did not find a large compressing artery near the aneurysm location. Therefore, the possibility of a crossing vessel as the cause for such aneurysm is less likely.

Phasic variations during the cardiac cycle. We found that the diameter of the coronary veins was greater during late systole compared with mid-diastole, which is consistent with a previous imaging study performed by Tada et al. (34). However, we did not observe a significant size difference between the 2 phases. Our data also showed that the CS contracts considerably during atrial systole. Therefore, we would recommend that all images throughout the cardiac cycle be evaluated during pre-procedural planning for a more thorough assessment of the coronary veins.

Study limitations. We cannot exclude the possibility that arrhythmias may develop in our patients with coronary venous aneurysm in the future. In the present report, there was no clear explanation of the origin of the aneurysmal abnormality in most of our patients. We also can not exclude the possibility of mild chronic tricuspid regurgitation, pulmonary hypertension with increased back pressure, or both as predisposing factors for the development of aneurysm in our patients. However, based on negative clinical history and the fact that the average diameter of normal veins in the groups with and without an aneurysm was not significantly different, this possibility is less likely. In addition, because the cases were analyzed in consensus, interobserver variability could not be evaluated.

CONCLUSIONS

Coronary vein aneurysms, especially fusiform dilatations, are not uncommon in patients without a history of cardiac arrhythmia and can be well visualized on routine coronary CTAs. It is important to realize that many of these focal dilatations may be incidental findings and may not result in cardiac arrhythmias.

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REFERENCES

1. McAlpine WA. Heart and Coronary Arteries: An Anatomical Atlas for Clinical Diagnosis, Radiological Investigation, and Surgical Treatment. New York, NY: Springer-Verlag, 1975: 188–96.
2. von Lüdinghausen M. The venous drainage of the human myocardium. Adv Anat Embryol Cell Biol 2003; 168:1–104.
3. Silver MA, Rowley NE. The functional anatomy of the human coronary sinus. Am Heart J 1988;115: 1080–4.
4. El-Maasarany S, Ferrett CG, Firth A, et al. The coronary sinus conduit function: anatomical study (relationship to adjacent structures). Europace 2005;7:475–81.
5. Meisel E, Pfeiffer D, Engelmann L, et al. Investigation of coronary venous anatomy by retrograde venography in patients with malignant ventricular tachycardia. J Am Coll Cardiol 2001; 104:442–7.
6. Saremi F, Krishnan S. Cardiac conduction system: anatomic landmarks relevant to interventional electrophysiologic techniques demonstrated with 64-detector CT. Radiographics 2007;27:1539–65.
7. Lemola K, Mueller G, Desjardins B, et al. Topographic analysis of the coronary sinus and major cardiac veins by computed tomography. Heart Rhythm 2005;2:694–9.
8. Jongbloed MRM, Lamb HJ, Bax JJ, et al. Noninvasive visualization of the cardiac venous system using multislice computed tomography. J Am Coll Cardiol 2005;45:749–53.
9. Ho SY, Russell G, Rowland E. Coronary venous aneurysms and accessory atrioventricular connections. Br Heart J 1988;60:348–51.
10. Gerlis LM, Davies MJ, Boyle R. Pre-excitation due to sinoventricular connexions associated with coronary sinus aneurysms. A report of two cases. Br Heart J 1985;53:314–22.

11. Sun Y, Arruda M, Otomo K, et al. Coronary sinus-ventricular accessory connections producing posteroseptal and left posterior accessory pathways: incidence and electrophysiological identification. Circulation 2002;106: 1362–7.
12. Giorgberidze I, Saksena A, Kroll RB, et al. Efficacy and safety of radiofrequency catheter ablation of left-sided accessory pathways through the coronary sinus. Am J Cardiol 1995;76: 359–65.
13. Willerson JT, Kereiakes DJ. Cardiac resynchronization therapy: helpful now in selected patients with CHF. Circulation 2004;109:308–9.
14. Loukas M, Tubbs RS, Jordan R. Aneurysm of the great cardiac vein. Surg Radiol Anat 2007;29:169–72.
15. Katritsis DG. The coronary sinus: passive bystander or source of arrhythmia? Heart Rhythm 2004;1:113–6.
16. Omran H, Pfeiffer D, Tebbenjohanns J, et al. Echocardiographic imaging of coronary sinus diverticula and middle cardiac veins in patients with preexcitation syndrome: impact on radiofrequency catheter ablation of posteroseptal accessory pathways. Pacing Clin Electrophysiol 1995;18:1236–43.
17. Schumacher B, Tebbenjohanns J, Pfeiffer D, et al. Prospective study of retrograde coronary venography in patients with posteroseptal and left-sided accessory atrioventricular pathways. Am Heart J 1995;130:1031–9.
18. Beukema WP, Van Dessel PF, Van Hemel NM, et al. Radiofrequency catheter ablation of accessory pathways associated with a coronary sinus aneurysm. Eur Heart J 1994;15:1415–8.
19. Guiraudon GM, Guiraudon CM, Klein GJ, et al. The coronary sinus diverticulum: a pathologic entity associated with the Wolff–Parkinson–White syndrome. Am J Cardiol 1988; 62:733–5.
20. Budoff MJ, Achenbach S, Fayad Z, et al., Society of Cardiovascular Computed Tomography, Society of Atherosclerosis Imaging and Prevention, Society for Cardiovascular Angiography and Interventions, American Society of Nuclear Cardiology. Task Force 12: training in advanced cardiovascular imaging (computed tomography): endorsed by the American Society of Nuclear Cardiology, Society for Cardiovascular Angiography and Interventions, Society of Atherosclerosis Imaging and Prevention, and Society of Cardiovascular Computed Tomography. J Am Coll Cardiol 2006;47: 915–20.
21. MacDevitt DT, Lohr JM, Martin KD, Welling RE, Sampson MG. Bilateral popliteal vein aneurysms. Ann Vasc Surg 1993;7:282–6.
22. Karaca M, Bilge O, Hakan Dinckal M, Ucerler H. The anatomic barriers in the coronary sinus: implications for clinical procedures. J Interv Card Electrophysiol 2005;14:89–94.
23. Corcoran SJ, Lawrence C, McGuire MA. The valve of Vieussens: an important cause of difficulty in advancing catheters into the cardiac veins. J Cardiovasc Electrophysiol 1999;10:804–8.
24. Van Meter C Jr, LeBlanc JG, Culpepper WS III, Ochsner JL. Partial anomalous pulmonary venous return. Circulation 1990;82 Suppl:IV195–8.
25. Davidson NC, Cooper MJ, Ross DL. Radiofrequency ablation of a posteroseptal accessory pathway associated with two diverticula of the coronary sinus. Circulation 2001;104:240–1.
26. Binder TM, Rosenhek R, Frank H, Gwechenberger M, Maurer G, Baumgartner H. Congenital malformations of the right atrium and the coronary sinus: an analysis based on 103 cases reported in the literature and two additional cases. Chest 2000;117: 1740–8.
27. Lev M, Saphir O. Endophlebohypertrophy and phlebosclerosis. I. The popliteal vein. AMA Arch Pathol 1951;51:154–78.
28. Schatz IJ, Fine G. Venous aneurysms. N Engl J Med 1962;266:1310–2.
29. von Lüdinghausen M, Ohmachi N, Boot C. Myocardial coverage of the coronary sinus and related veins. Clin Anat 1992;5:1–15.
30. Cabrera JA, Sánchez-Quintana D, Ho SY, et al. Angiographic anatomy of the inferior right atrial isthmus in patients with and without history of common atrial flutter. Circulation 1999;99:3017–23.
31. Saremi F, Pourzand L, Krishnan S, et al. Right atrial cavotricuspid isthmus: anatomic characterization with multidetector row CT. Radiology 2008; 247:658–68.
32. Bales GS. Great cardiac vein variations. Clin Anat 2004;17:436–43.
33. Pejkovic B, Bogdanovic D. The great cardiac vein. Surg Radiol Anat 1992; 14:23–8.
34. Tada H, Kurosaki K, Naito S, et al. Three-dimensional visualization of the coronary venous system using multidetector row computed tomography. Circ J 2005:69:165–70.

Key Words: coronary vein aneurysm ■ coronary computed tomography angiography ■ cardiac arrhythmias ■ ventricular pre-excitation ■ accessory pathways.